sis. Indeed this is borne out by the prevalence figure for the Western Isles of Scotland[2] of only 81 per 100 000, which is one of the lowest prevalence rates found in the United Kingdom and yet these islands have the highest percentage of Scottish surnames.

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1 Rothwell PM, Charlton D. High incidence and prevalence of multiple sclerosis in south east Scotland: evidence of a genetic predisposition. *J Neurol Neurosurg Psychiatry* 1998;**64**:730–5.
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High incidence and prevalence of multiple sclerosis in south east Scotland: evidence of a genetic predisposition

We read with interest the results of Rothwell and Charlton regarding the incidence and prevalence of multiple sclerosis in south east Scotland.[1] They have identified standardised multiple sclerosis prevalence rates for the Lothian and Border Regions of 203 and 219 per 100 000 respectively, the results challenging the theory that the high prevalence rates previously reported in Scotland are peculiar to the north east and its offshore islands. The authors postulate that the apparent step in prevalence rates between England and Scotland may be due to the distinctive Celtic ancestry of the Scottish population as can be crudely measured by surnames prefixed with Mc or Mac.

In Northern Ireland we have also identified a much higher prevalence rate for the disease than exists in England and Wales and have speculated that the similar rate to that in Scotland is at leastly partly a function of the common ethnic origins of the two populations.[2] The contiguous region of Coleraine, Moyle, Ballymena, and Ballymoney lies less than 20 miles from Scotland at its closest point and has a standardised prevalence rate for all multiple sclerosis, based on the 1961 census population for Northern Ireland,[3] of 230 (95% confidence interval (95% Cl) 207–256) per 100 000. Using a similar method to Rothwell and Charlton (British Telecom phone book of the area), 17% of the study population had a surname prefixed with Mc or Mac and it is of note that 22.9% of prevalent cases had such a surname prefix (odds ratio = 1.46, 95% Cl 1.09–1.93, χ^2=6.82, p=0.009).

Our results support the conclusion of Rothwell and Charlton that Celtic ancestry is a risk factor for multiple sclerosis and confirm the existence of a step in multiple sclerosis prevalence in the British Isles between England/Wales and Scotland/Northern Ireland.

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1 Rothwell PM, Charlton D. High incidence and prevalence of multiple sclerosis in south east Scotland: evidence of a genetic predisposition. *J Neurol Neurosurg Psychiatry* 1998;**64**:730–5.
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The author's reply:

The study of the prevalence of multiple sclerosis in Northern Ireland by McDonnell and Hawkins is interesting.[1] The findings are similar to those of the recent study in south east Scotland.[2] Both studies suggest that there is an increased prevalence of multiple sclerosis in the north of the British Isles compared with the south. It seems likely, as McDonnell and Hawkins suggest, that this at least partly reflects differences in the genetic susceptibility of the respective populations.

The south east Scotland study did, as Shepherd suggests, attempt to link the high prevalence of multiple sclerosis to Scottish ancestry. However, the study used a standard text of several hundred surnames which are considered to have originated in Scotland,[3] rather than just those prefixed with Mc or Mac. This is obviously still a very crude approach to the problem, but any bias is likely to have weakened rather than strengthened the association. The proportion of cases in the telephone book with a surname pre-fixed with Mc or Mac was simply used as a crude illustration of the fact that the differences in apparent ancestry between the Scotland and England are still considerable. This is supported by major differences in the HLA types of the two populations.[4] Contrary to Shepherd's assertion, the Highlands and Islands telephone book does include Orkney. However, he is correct to point out that the prevalence of surnames pre-fixed with Mc or Mac is indeed lower on Orkney than in the region as a whole.

Further insights into the high prevalence of multiple sclerosis in the north of the British Isles might come from a prevalence study which is currently being planned on the Isle of Skye.

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Albendazole therapy for subarachnoid cysticerci: clinical and neuroimaging analysis of 17 patients

By contrast with the weaknesses of anecdotal observations from case series, the power of randomised clinical trials for deciding the benefit of therapy has become increasingly evident and indisputable world wide. Nowadays, to argue against the validity of this assertion may seem superfluous; however, a recent paper reported by Del Brutto[1] regarding treatment in neurocysticercosis ignores basic procedures for well performed clinical trials by using inappropriate and misleading methodology to evaluate medical therapy.

By definition, a clinical trial is a prospective study comparing the effect and value of treatment against a control in human subjects. The main drawback of Del Brutto's report is that it does not include a control group against which the intervention group is compared; therefore, its results are definitely flawed. Additionally, a basic experimental study design requires at least minimal information regarding inclusion and exclusion criteria, randomisation, and definitions of response or outcome variables. This information is not provided by Del Brutto's report; its design fails to protect against potential bias in patient selection or evaluation of outcome. The definition of subarachnoid cysterci used by Del Brutto was based on "appearance on CT of hypodense cystic lesions located over the convexity of the cerebral hemispheres, the sylvian fissure, or the CSF cisterns at the base of the brain". It is well known that there are many other diagnostic possibilities to be considered in the differential diagnosis of subarachnoid hypodense lesions.[2 3] Besides, CT is not a reliable procedure for diagnosing subarachnoid cysterci, as is MRI. In fact, we cannot be completely sure, for example, that the CT images shown in the report of Del Brutto correspond to subarachnoid cysterci. If we were to use MRI on this patient, they might correspond to a parenchymal cyst which resolved as a reflection of the natural history of the condition. There is no evidence that objectively confirms or rejects this assertion.

Del Brutto's report[1] maintains that evaluation of the therapeutic response to albendazole included comparison of the size of the cysts, as well as clinical evaluation of patients before and after treatment. To consider the size of cysts as a response variable is certainly useless because of the obvious difficulties in measuring cyst size in the subsequent follow up CT. It is also widely accepted that the clinical manifestations of neurocysticercosis are polymorphic, and their clinical course is unpredictable; therefore, the clinical manifestations as an outcome variable is entirely biased. Another personal appreciation of Del Brutto[1] is that albendazole reaches high concentrations in CSF, and has been used with success in some patients with subarachnoid cysts; nevertheless, studies used as support[1] of this presumption are similarly flawed in that they are not randomised or blinded, having historical control groups or patients who served as their own control, and regarding clinical evaluation as an outcome variable.

Whereas it is generally assumed that albendazole is effective treatment for neurocysticercosis, a critical review of the literature[3 4] suggests that the studies on which these assumptions are based are defective in terms of patient selection, assignment to treatment, and selection and measurement of outcome variables. Many authors have warned that this therapy in some patients might sometimes be harmful, particularly in the subarachnoideal localisation, because some patients have developed arteritis and hydrocephalus after the administration of antihelminthic drugs.[3] According to these authors a parasite may be easily removed surgically at a cystic stage before an inflammatory reaction develops.[3] A randomised clinical trial of treatment of neurocysticercosis[4] considers the question of to what extent and in which patients treatment with either praziquantel or albendazole is effective. The improvement attributed to these drugs in several studies may be related to the lack of appropriate controls and is likely to be a reflection of the natural history of the condition. The authors point